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Secondary Sulfate Effects?

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Maynard et al. (2007) related mortality with ambient particulate black carbon (BC) and sulfate (SO_4). They also associated SO_4 in Boston, Massachusetts, with "secondary," distant "coal combustion." Their correlation of 0.44 between BC and SO_4, however, suggests the importance of local source(s) of SO_4, including vehicle exhaust (especially diesel).

Centrally monitored BC poorly characterizes exposure to traffic emissions and results in exposure misestimation, causing distorted or undetected health associations (Ito et al. 2004). Maynard et al. (2007) address this deficiency by geocoding BC estimates to a decedent's residence; a central monitor characterizes SO_4 exposure. In their single-pollutant models, BC and SO_4 are associated with daily mortality, but in multipollutant models, only BC retains significance.

The association of mortality with SO_4 is inconsistent. In a review of toxicologic studies, Schlesinger and Cassee (2003) suggested that SO_4 is benign. In an *in vivo* study of $PM_{2.5}$ (particulate matter ≤ 2.5 μm in aerodynamic diameter) components, Seagrave et al. (2006) found that lung toxicity and inflammation correlated with vehicular but not secondary particles, including SO_4. However, vehicular emissions are consistently associated with cardiac or other end points (Grahame and Schlesinger 2007). How does secondary SO_4 cause mortality, if harmful biological mechanisms have not been found? Or could the mortality association with SO_4 of Maynard et al. (2007) be linked with other local PM sources?

Maynard et al. (2007) cited mortality associations for coal and traffic tracers reported by Laden et al. (2000), but other study findings differ. In a reanalysis, Schwartz (2003) found that their traffic variable remained significant, but their regional "coal" variable became insignificant. We (Grahame and Hidy 2004) noted that among the six cities described by Laden et al. (2000), Boston had the lowest levels of the "coal tracer" (selenium); only Boston had a significant association with an apparent coal source. Similarly, only in Boston was an association with SO_4 significant. Finding that local residual oil sources emitted over half the Se and SO_4 in Boston, these authors (Grahame and Hidy 2004) concluded from toxicology that these results represented effects of residual oil emissions.

SO_4 is elevated near major roadways. Reponen et al. (2003) showed an SO_4 gradient that declined based on distance from a midwestern freeway; Riediker et al. (2004) found that among highway-related factors, only the "speed changing" factor, which included emissions from accelerating diesel engines, showed elevated SO_4. These findings are relevant to the monitoring conditions of Maynard et al. (2007). Other Boston studies show commingling between SO_4 and vehicular indicators. Clarke et al. (2000), for example, reported that loadings of BC are as high in an SO_4 indicator as in the traffic indicator. Because coal plants emit virtually no BC (Edgerton E, Mueller P, Monroe L, Jansen J, Waid C, unpublished data), high BC in urban SO_4 indicators reflects both SO_4 and BC from diesels in an SO_4 factor, derived from measurements near a thoroughfare. The SO_4–BC correlation described by Maynard et al. (2007) thus suggests that SO_4 associations reflect statistical comingling of vehicular and coal emissions.

Association of harm from traffic emissions, but not SO_4 (or secondary aerosols generally), has been found in panel and epidemiology studies that *a*) precisely measure exposure to local vehicular emissions, and *b*) test effects of secondary $PM_{2.5}$–SO_4 compared with local emissions. Health end points in such studies include long-term mortality, heart rate variability reduction, ST-segment depression, cardiac effects, vasoconstriction, increased blood pressure, or morbidity [reviewed by Grahame and Schlesinger (2007)]. Kodavanti et al. (2005) found no effects only with the highest SO_4 concentrations.

We suggest that findings of harmful exposure to secondary SO_4 per se are tenuous until physiologic mechanisms are identified that support toxicity near ambient concentrations.

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Secondary Sulfate Effects: Schwartz Responds

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I am not convinced by the argument of Grahame and Hidey in their letter. In fact, I find it unsupportable by modern atmospheric science and statistics. First, they argue that much of the sulfates in Boston, Massachusetts, derive locally, rather than from long-range transport, and that diesel exhaust is a probable major source. Sulfates, a major source of fine particles in Boston and other East Coast communities, derive primarily from coal-burning power plants, and this clearly may be uncomfortable for the electric utility industry and the Department of Energy; however, it is

equally clear that there is little doubt about it among atmospheric scientists. For example, Salmon et al. (1997) reported that sulfate concentrations at a monitoring station in a limited-access conservation area in a rural part of western Massachusetts were almost identical to the sulfate concentrations in Boston, whereas the elemental carbon concentrations, which are markers of traffic, particularly the diesel exhaust Grahame and Hidy postulate as the source of sulfates in Boston, were quite different. The sulfate levels in Boston cannot simultaneously be driven by local traffic and still be identical to those in a distant rural area.

This similarity in sulfates persists to later periods. The U.S. Environmental Protection Agency's Technology Transfer Network (U.S. EPA 2007) provides data on sulfate levels measured on the same days in Springfield, Massachusetts, at the western end of the state, and Boston, 95 miles to the east (Table 1). Apparently, truck traffic is almost identical in these cities.

Table 1. Sulfate concentrations by year in Springfield and Boston, Massachusetts.

Year/city	No. of days	Mean (μg/m^3)
2001		
Boston	117	3.08
Springfield	117	3.04
2002		
Boston	122	2.98
Springfield	122	2.51
2003		
Boston	124	3.03
Springfield	124	2.00
2004		
Boston	125	3.08
Springfield	125	3.01
2005		
Boston	106	3.19
Springfield	106	3.17

Hourly sulfate monitors are operated in St. Louis, Missouri, Boston, and elsewhere. Hourly concentrations peak in the afternoon after the boundary layer has broken up, and transported sulfates can mix down to ground level. In contrast, concentrations of BC, a marker of diesel exhaust, peak near 0600 hours.

Regarding Grahame and Hidy's second point, in our study (Maynard et al. 2007) we reported that the sulfate effect was no longer significant when we controlled for BC. However, because there were far fewer days with sulfate and because the coefficient was similar to the coefficient described by Laden et al. (2000), we stated that our finding strengthened the evidence for an association with mortality. We see nothing in Grahame and Hidy's letter to lead us to change that view. Yes, the association is not statistically significant. But judgments about associations are made by combining evidence across studies, not on single studies. And in a meta-analysis, adding a study with a positive coefficient of similar size to previous studies increases the significance of the association when the cities are all combined. Thus our statement that this result adds to the support for a sulfate association remains the most reasonable interpretation. Finally, Grahame and Hidy make much of the change in effect size for sulfate when controlling for BC. Although this does indicate that there is some correlation of the measures, this correlation is to be expected, because common weather patterns drive some of the day-to-day variation in most pollutants. It does not imply that sulfate in Boston comes to a substantial extent from diesel engines.

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